**Shock**

It is a systemic state of low tissue perfusion which is inadequate for normal cellular respiration. With insufficient delivery of oxygen and glucose, cells switch from aerobic to anaerobic metabolism. If perfusion is not restored in timely fashion, cell death occur.

**Classification of shock**
- Hypovolemic
- Cardiogenic
- Obstructive
- Distributive
- Endocrine

**Hypovolemic shock**
It is caused by reduced circulating volume. It may be due to haemorrhagic or non hemorrhagic causes. Non hemorrhagic causes include poor fluid intake, and excessive fluid loss due to vomiting, diarrhoeae, urinary loss as in diabetes, evaporation and third space loss. This type of shock also include traumatic shock and burn shock.

**Cardiogenic shock**
It is due to primary failure of the heart to pump blood to the tissues. Causes include MI, arrhythmia, valvular heart disease, blunt myocardial injury and cardiomyopathy.

**Obstructive shock**
Here there is reduction in preload due to mechanical obstruction of cardiac filling. Common causes include cardiac tamponade, tension pneumothorax, massive pulmonary embolism and air embolism. In each there is reduction in the filling of the left and or right sides of the heart leading to reduced preload and a fall in cardiac output.

**Distributive shock**
It describes the pattern of cardiovascular responses to characterizing a variety of conditions including septic shock, anaphylactic shock, spinal cord injury, vasovagal shock and psychogenic shock. Inadequate organ perfusion is accompanied by vascular dilatation with hypotension, low systemic vascular resistance, inadequate afterload and a resulting abnormally high cardiac output.

**Endocrine shock**
It may present as a combination of hypovolemic, cardiogenic, and distributive shock. Causes of endocrine shock include hypo and hyperthyroidism and adrenal insufficiency. Hypothyroidism causes a shock state similar to that of neurogenic shock as a result of disordered vascular and cardiac responsiveness to circulating catecholamines. Cardiac output falls because of bradycardia in low inotropy.
Thyrotoxicosis may cause a high output cardiac failure. Adrenal insufficiency cause shock as a result of hypovolemia and a poor response to circulating and exogenous catecholamine.

**Septic (endotoxic) shock (Pathophysiology)**

**Hyperdynamic (warm) septic shock.** This occurs in serious Gram-negative infections, for example from strangulated intestine, peritonitis, leaking oesophageal or intestinal anastomoses, or suppurative biliary conditions. At first, the patient has abnormal or increased cardiac output with tachycardia and a warm, dry skin, but the blood is shunted past the tissue cells, which become damaged by anaerobic metabolism (lactic acidosis). The capillary membranes start to leak and endotoxin is absorbed into the blood-stream, leading to a generalised systemic inflammatory state. The immediate and ready treatment of the cause, including the drainage of pus, is vital to the recovery of the patient at this stage (in strangulated hernia ‘the danger is in the delay, not in the operation’).

**Hypovolaemic hypodynamic (cold) septic shock.** This follows if severe sepsis or endotoxaemia is allowed to persist. Generalised capillary leakage and other fluid losses lead to severe hypovolaemia with reduced cardiac output, tachy-cardia and vasoconstriction. The systemic infection induces cardiac depression, pulmonary hypertension, pulmonary oedema and hypoxia which, in turn, reduce cardiac output still further. The patient becomes cold, clammy, drowsy and tachypnoeic, but still can be converted to hyperdynamic (warm) shock by the administration of several litres of plasma or other colloidal solution. The similar use of crys-talloid solutions may give rise to systemic and pulmonary oedema because of the larger volumes necessary.

**Hypovolaemic**
- Blood loss (ruptured abdominal aortic aneurysm, upper GI bleed, multiple fractures, etc.).
- Plasma loss (burns, pancreatitis).
- Extracellular fluid losses (vomiting, diarrhoea, intestinal fistula).

**Cardiogenic**
- Myocardial infarction.
- Dysrhythmias (AF, VT, AFLutter).
- Pulmonary embolus.
- Cardiac tamponade.
- Valvular heart disease

**Septic**
Gram –ve or, less often, Gram +ve infections.

**Anaphylactic/distributive**
Release of vasoactive substances when a sensitized individual is exposed to the appropriate antigen.

**Clinical features**

**Hypovolaemic and cardiogenic**
- Pallor, coldness, sweating and restlessness.
- Tachycardia, weak pulse, low BP and oliguria.

**Septic**
- Initially warm, flushed skin and bounding pulse.
- Later confusion and low output picture.
Complications
• 'SIRS' may ensue if shock not corrected.
• Acute renal failure (acute tubular necrosis).
• Hepatic failure.
• Stress ulceration.

Management

Resuscitation
Immediate resuscitation for patients presenting in shock are to ensure patent air way and adequate oxygenation and ventilation. Once air way and breathing are assessed and controlled, attention is directed to cardiovascular resuscitation.

Resuscitation includes:
• Fluid therapy
• Vasopressors and inotropic support

Monitoring for patients with shock

Minimum

• Monitor pulse, BP, temperature, respiratory rate and urinary output.
• Establish good i.v. access
• ECG, cardiac enzymes, echocardiography.
• Hb, Hct, U+E, creatinine.
• Group and crossmatch blood: haemorrhage.
• Blood cultures: sepsis.
• Arterial blood gases.
• ECG
• Pulse oxymeter
• Urine output

Additional modalities
• Set up CVP line (possibly Swan–Ganz catheter as well).
• Cardiac output
• Base deficit and serum lactate

Note
Deal with the cause of the shock: e.g. stop the bleeding, drain the abscess, remove the source of the anaphylactic antigen, etc.

Notes on terms used
Resistance arterioles are the small-calibre vessels, 0.02—0.05 mm in diameter, containing abundant smooth muscle in their walls, the tone of which is controlled by local humoral factors and the sympathetic nerve fibres. The calibre of these small vessels gives rise to the peripheral vascular resistance, controlling blood pressure and blood flow through the capillary beds. The larger arteries merely serve to supply the arterioles with blood.
Capacitance veins comprise the entire venous network from the postcapillary venules to the large-calibre veins in limbs, abdomen and thorax and which normally contain 70 per cent of the circulating blood volume. Although thin walled with relatively little smooth muscle, sympathetic nerve stimulation contracts them, reducing their diameter and emptying the blood into the arterial side of the circulation.

A colloidal solution is one in which the majority of solute particles has a molecular weight greater than 30 000. The term includes all plasma solutions, including human plasma protein fraction (HPPF), dextrans, gelatin (e.g. Haemaccel) and hydroxyethyl starch. Blood is not usually included in this term.

Minute volume ventilation is the volume of air (or oxygen) which enters the patient’s lungs in 1 (each) minute, and is the product of respiratory rate and tidal volume.

Hyperventilation occurs when the patient is ‘overbreathing’ due to pain, anxiety or shock, such that the arterial carbon dioxide tension (PaCO2) is lowered from the normal 40 mmHg (5.5 kPa).

Aspects of the pathophysiology of haemorrhage and shock
Low cardiac output is an early feature in shock, except for warm septic shock and neurogenic shock. Vasconstriction occurs in an attempt to maintain perfusion pressures to the vital organs, such as the brain, liver and kidneys, as well as the heart muscle itself. Venoconstriction pushes more blood into the dynamic circulation whilst tachycardia helps to maintain a falling cardiac output. The minute ventilation rises 1.5—2 times and the respiratory rate 2—3 times maintaining oxygenation (except in cardiogenic shock with pulmonary oedema). The renal blood flow is reduced with consequent reduction in glomerular filtration and urine output. The renin—angiotensin mechanism is activated with further vasoconstriction and aldosterone release, causing salt and water retention. Release of antidiuretic hormone (ADH) decreases the volume and increases the concentration of urine. However, in early sepsis the patient, although hypovolaemic, may produce inappropriately large amounts of dilute urine.

As cardiac output falls, the hypotension and tachycardia cause poor perfusion of the coronary arteries, and this, in conjunction with hypoxia, metabolic acidosis and the release of specific cardiac depressants (endotoxaemia or pancreatitis), causes yet further cardiac depression and pump failure. The cells become starved of oxygen, and anaerobic metabolism leads to lactic acidosis. Eventually, the cell membranes cannot pump sodium out of the cells; sodium enters the cells and potassium leaks out. Thus, the serum potassium is elevated. Calcium, however, leaks into the cells lowering the serum calcium. Furthermore, the intracellular lysosomes break down and release powerful enzymes causing further damage — ‘the sick cell syndrome’. The platelets are activated in shock owing to the stagnation of blood in the capillaries. Blood sludging with red cell aggregation may progress to the formation of small clots and, indeed, to DIC. Several coagulation factors are consumed (platelets, fibrinogen, Factor V, Factor VIII, prothrombin), and troublesome bleeding may occur from needle puncture sites, wound edges and mucosal surfaces.
Diagnosis
The prognosis of a shocked patient is related to the duration and degree of the shocked state, therefore prompt diagnosis of the type of shock is essential. It should be remembered that a thready and irregular pulse can make the measurement of blood pressure inaccurate and misleading. Intra-arterial pressure monitoring should be used. The ECG should be monitored to detect any arrhythmias that may occur. A chest X-ray may reveal mediastinal trauma or cardiac tamponade.

Central venous pressure
The measurement of central venous pressure (CVP) and its response to a small fluid challenge (200 ml of crystalloid or colloid) may assist in distinguishing between cardiogenic shock and hypovolaemic shock, but it must be emphasised that, in the seriously ill patient, the CVP is not a reliable indicator of left ventricular function because of the wide disparity that can exist between the left and the right ventricular functions.

Pulmonary capillary wedge pressure
The pulmonary capillary wedge pressure (PCWP) is a better indicator of both circulating blood volume and left ventricular function. PCWP is obtained by a pulmonary artery flotation balloon catheter (Swan—Ganz). This can be used to differentiate between left and right ventricular failure, pulmonary embolus, septic shock and ruptured mitral valve, and can also be an accurate guide to therapy with fluids, inotropic agents and vasodilators. It may also be used to measure cardiac output by a thermodilution technique simply at the bedside.

Types of shock and their management

Hypovolaemic shock
- **Causes:** trauma, ruptured AAA, ruptured ectopic, postoperative haemorrhage, profound dehydration, burns, pancreatitis.
- **Clinical features.** As above with history of trauma/surgery/illness.
- **Treatment.**
  - Lie patient flat; high flow O$_2$; lift legs to autotransfuse if no IV access.
  - Repeat fluid infusion 500mL IV rapidly: you should see rise in BP.
  - Take blood and send for FBC, U & E, clotting, and cross-match.
  - Take arterial blood gas: estimate Hb, K as well as blood gases.
  - Treat K$^+$.  
  - If no rapid improvement in BP look for other causes.

Anaphylactic shock
- **Causes:** drug allergy, blood product reaction, latex allergy.
- **Clinical features:** history of sudden onset after administration of drug. Stridor or bronchospasm, angioedema, urticaria, pruritus, rash.
- **Treatment.**
  - Sit patient up; give high flow O$_2$; call anaesthetist if stridor.
If IV access: give 1mL of 1:10 000 adrenaline bolus; flush; then 100mg hydrocortisone bolus; flush; then 10mg chlorpheniramine IV.

Repeat again in 5-10 min if no improvement.

If no IV access: give 1mL 1:1000 adrenaline IM. Then secure IV access.

If wheezy give 5mL nebulized salbutamol.

Septic shock

- **Cause.** Overwhelming sepsis.
- **Clinical features.** May be the same as hypovolaemic shock or, if established, with circulatory collapse. Earlier in the evolution the patient may look septic pyrexial, flushed, bounding pulses.
- **Treatment.**
  - As for hypovolaemic shock.
  - Take blood cultures; then give IV cefuroxime 750mg tds.

Cardiogenic shock

- **Rapidly reversible causes:** cardiac tamponade (trauma, post-cardiac surgery), arrhythmias, tension pneumothorax.
- **Other causes:** fluid overload and CCF, MI, PE, SBE, aortic dissection, decompensated valvular heart disease.
- **Clinical features.** History of recent surgery/trauma, chest pain, dyspnoea, palpitations.

- **Treatment.**
  - Give high flow O\textsubscript{2}.
  - Give 2.5mg morphine IV (anxiolytic, venodilator, analgesic, anti-arrhythmic).
  - Put patient on cardiac monitors; request 12-lead ECG.
  - Treat arrhythmias.
  - Treat myocardial ischaemia with 0.1mg GTN, 300mg aspirin.
  - Auscultate heart sounds and lung fields.
  - Treat tension pneumothorax, cardiac tamponade.
  - Consider central venous and peripheral arterial monitoring.
  - Send blood for arterial blood gases, FBC, U & E, clotting, troponin.
  - Catheterize the patient.
  - Request CXR look for pulmonary oedema.
  - Treat fluid overload with diuretics: frusemide 40mg IV.
  - Consider transthoracic echo to exclude pericardial effusion and valvular lesions, and to assess LV function.